VOL. 35. MAY, 1908. No. 5.

The Journal
OF
Nervous and Mental Disease

Original Articles

A CASE OF MENINGEAL TUMOR COMPRESSING THE CEREBELLUM

By Helen Baldwin, M.D.,
PHYSICIAN TO THE NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

The symptoms of brain tumors, especially of rapidly growing neoplasms, are often so definite, so obvious and so distressing that they demand recognition and make a diagnosis relatively easy. With slowly growing tumors, however, the case is different and these may attain a large size without causing any one of the classical symptoms of new growths in the brain—headache, vomiting or optic neuritis.

The patient whose history is here given developed the first symptoms of her disease ten years before her death, the tumor grew to such a size that it would seem that it must have given characteristic symptoms, yet optic neuritis never developed, there were only a few attacks of vomiting throughout the illness, and these were evidently due to acute indigestion, and while headaches occurred for short periods daily in the early years of the disease, during the three years before the patient's death they did not recur. During the course of the illness the symptoms so closely simulated those of Parkinson's disease, of arteriosclerosis of the vessels in the cerebrum and cerebellum, and of cyst of the cerebellum, that while the question of brain tumor was always considered, the symptoms at no time seemed to justify an exploratory operation.

The first symptoms directly referable to the tumor were noticed ten years before the fatal termination of the disease. The

patient on waking one morning was seized with a very severe pain over the suboccipital region of the brain. This lasted only a few moments, but recurred each succeeding morning on waking. At about this time, when walking one day, she suddenly fell with no apparent cause, with no vertigo, and with no loss of consciousness.

The patient was at this time 54 years of age, a married woman who had borne three children. Her father died suddenly of

FIG. 1. Showing the Position of the Tumor.

heart disease. Her mother died of uremia, following an operation for pelvic tumor. The patient had ten brothers and sisters, all but two of whom survived her.

The early history of her sickness can best be described in her own words. This record was written in the fall of 1903.

. . . I do not believe that any one ever had such good health, certainly not any better, than I enjoyed until the age of 54 years. During this period, I sustained many trials, some of a nature peculiarly calculated to break down the nervous system of women, but I did not break down. Indeed I often reflected with pleasure that not only my muscles, digestive apparatus, etc., were always in perfect running order, but my brain was always singularly clear and buoyant. It seemed to me often as if I lived in a glass house on the summit of a lofty mountain where I could see in every direction an almost illimitable distance looking through an atmosphere of blue and gold. The delight I experienced in the clearness of this view was immense. On account of it I was never conscious of depression or of irritation for more than a few moments at a time. I lived in an equable golden calm as in a sunrise or sunset cloud. I emphasize this habitual condition because it was on account of it that the first symptoms of the present illness became so conspicuous from contrast and attracted my attention, as otherwise they might not have done.

In the winter of 1896, . . . on waking one morning I experienced a very sharp pain running transversely just below the occiput. It lasted between three and five minutes, then disappeared, and was heard no more of throughout the day. But the next morning at precisely the same time the suboccipital pain returned with precisely the same characters, and lasting precisely the same length of time. From this date, the same thing happened every morning for four years, and the pain never occurred at any other time of day. But in 1900 it did begin to come on occasionally at other times, always, however, lasting such a short time, three to five minutes, that it did not seem to me deserving of much attention, however severe it was while it lasted. Finally, in the year 1900, the morning pain instead of disappearing persisted and increased in severity, in extent, and territory, became complicated with nausea, then vomiting—assumed in fact all the characters of an ordinary sick headache.

I think I had never had a sick headache before. It lasted from early morning until early in the afternoon, then died away under the influence of phenalgine. This sick headache recurred every six months during the next year and a half. In the interval the head was as clear as usual, only the sharp attack of suboccipital pain continued to occur for five minutes every morning.

In June of 1901, I joined a party in an expedition to the Yellowstone Park, where I spent a week. On the first day, and after the thirty-six hours' railroad ride, I had a sick headache with moderate pain, but much nausea. The next day, and for a week, I was perfectly well. But the day before leaving I indulged in a hot bath from the geyser water and was seized in the night with an extremely violent pain in the head, not limited

as usual to its posterior third, but extending all over, and soon accompanied by retching and vomiting. These symptoms were so severe in the morning that I was quite unable to rise and accompany the party home. I remained in bed all day, took phenalgine, and gradually recovered. The following winter, however, I noted a gradual increase in the head symptoms occurring on waking in the morning, also a great difficulty in arising from a recumbent to a sitting position. Frequently there was an attack of nausea, and even vomiting, after getting out of bed. Great irritability of the bladder at this time, and that also frequently continued during the day, but altogether the first few hours in the morning were always pretty miserable. Nevertheless, I managed to do my work, usually after 10 A. M., and felt pretty well, but during the winter of 1901–1902, the attacks of sickness became more frequent, and towards spring about once every two or three weeks I was laid up in bed all day. At the end of May, I had an especially severe attack, which was arrested this time by nitroglycerine. I was then ordered to bed for a fortnight, the first such experience in my life, during which time I felt quite ill. I continued to take a few tablets of nitroglycerine daily.

On the seventh of June, I went into the country with my family, and stayed there until the seventh of October. Three days before I went to bed I suddenly lost in great measure my power of walking. I was walking home, . . . when it suddenly seemed to have become almost impossible to drag one foot after another. It was with great difficulty that I climbed up the steps to the house. This was the first of June, and from that date to the present my walking power has been greatly reduced. During the summer I at first only attempted to walk a few steps off the piazza. After a month I could sometimes walk a quarter of a mile, and in three months I could occasionally walk half a mile. There was no pain or stiffness in the legs at the time, but since the last week I have noticed, with some concern, that a great feeling of heaviness and dragging is liable to come on in the end of the back, and especially after walking. I cannot now walk more than from three to six blocks. At the time of this limitation in extent of walking capacity, I began to totter somewhat in walking. On going downstairs there was a tendency to pitch forwards. This tottering has not increased, but it has persisted. Since the last two months, I cannot walk more than three or four blocks, and that with the aid of a cane and an assistant's arm. From time to time I have fallen suddenly—not when out of doors, most frequently upon arising after sitting for a long time, perhaps especially in the evening. I would fall to the floor, and experience considerable difficulty in getting upon a chair. The fall was unaccompanied by either vertigo, giddiness or pain. Indeed no different sensation in any part of the body; the legs

simply gave way as if I had been on skates. After a moment or two, I could climb to my feet again and felt none the worse for the adventure. These attacks of falling have occurred about once in three or four weeks during the winter.

I do not find any symptoms of paralysis in any of the four limbs, either of motility or sensibility. Neither are there cramps, contracture of muscles, or stiffness in them. I can climb in and out of a bath tub usually with ease, though occasionally I need the maid to assist me, as also in arising from the bed in the morning. There is no steadiness or regular progression in this nor in any of the other symptoms. There is a constant sense of general fatigue and inability for exertion.

A slight tremor has occurred in my right hand during the last winter. It comes during repose and is scarcely rhythmical; its excursion is very small; it is not developed during motion or exertion. During the last two or three weeks, I think this tremor has been much less, and often for many hours it would be entirely absent. There is no fibrillary tremor.

A change in mental condition began in a subtle manner about six years ago. I began to lose the initiative, which had formerly been so active with me. I was not at all depressed or melancholy, but became relatively indifferent. It seemed as if a fine gauze veil were thrown over all the objects in which I had formerly been so intensely interested. It was like the life after death as the Greeks understood it when they described Hades. My interest in contemplation persisted, and even increased, but I acquired an increasing reluctance to effort and voluntary mental exertion. I appreciated Schopenhauer's "Condemnation of the Will," and felt confirmed in my view that his entire theory sprang from a deep inner consciousness of personal weakness of volition. This impressed me the more from its sharp contrast with the vivacity and strength of volition which had been a leading characteristic with me all my life. There was a facility of fatigue after mental exertion, quite comparable to that after walking. This became marked at the same time with the latter, that is after June, 1903, although the sense of loss of initiative had begun, as I have said, six years before. In the last week I have had for the first time a dragging heaviness in my left arm, and some stiffness when I move it backward.

In addition to the above symptoms recorded by the patient, there were others occurring at this time which were noted by her friends. These were mainly symptoms of ataxia. When walking about the house she would place her hands upon objects to support her or more often to regain her balance. When on the street she was inclined to walk with a broad base and with short

steps as though to steady her gait. When walking quickly she had a propulsive gait. If she wished to turn in her course she would stop abruptly and bend her body backward to regain her poise before turning. This propulsion was noted even five years before her death. In the latter part of this period it would frequently happen that if she turned out the light and the room became dark she would fall to the floor and be unable to rise

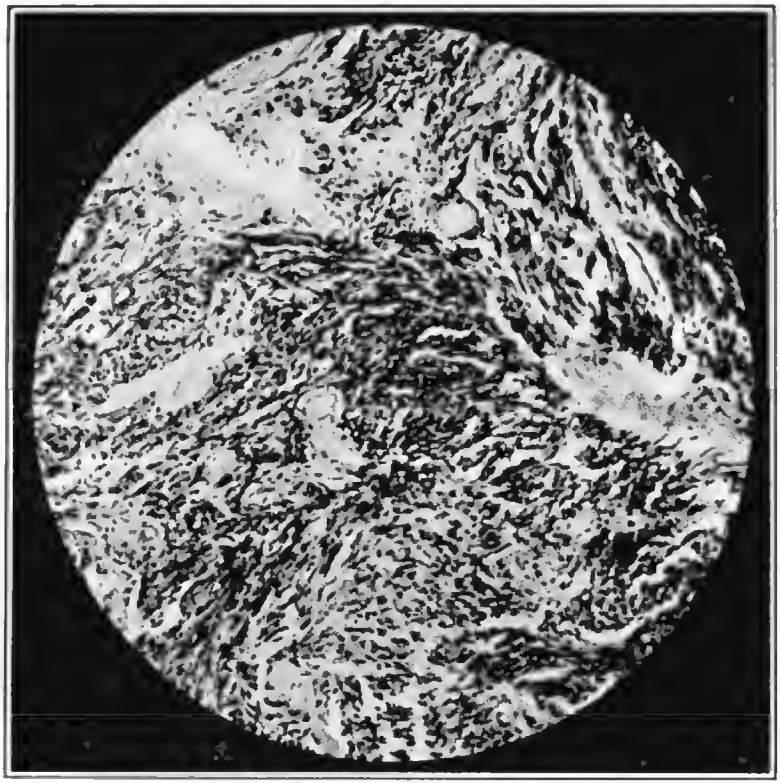

FIG. 2. Section of the Tumor.

until she had crept on her hands and knees to some object which she could use as a support. At this time also a tendency to drowsiness was becoming marked, and yawning with or without apparent sleepiness was a conspicuous symptom. After talking or laughing she would sometimes put her hand to the back of her neck as if in pain.

Three years before her death the patient came under the care of the writer. At this time the symptoms closely simulated those of Parkinson's disease, the gait being especially characteristic. She would start to walk with a resolute erect poise, but after a few steps she would begin to bend forward, and bending more and more, would hurry to reach some support. She had at this time a tremor of the right hand which with effort she could control for a time. She had also a slight tremor in her tongue. There was an immobility of countenance and an increasing slowness of mental processes. The fingers were held in a flexed position, so that at one time slight sores developed on the palmar surface of the right hand. The reflexes were normal. There was a general muscular weakness, more marked on the right side, but with no rigidity. When walking she had a tendency to fall to the right side. Sensation to touch, pressure and temperature over the skin seemed to be unimpaired. But she early lost the sense of taste and of touch within the mouth, so that she could not tell whether she was holding food in her mouth or not. There was at no time any paralysis of the palate. There was a weakness in the action of the epiglottis, so that frequently, while eating, particles of food or drink would pass into the larynx and choke her. Throughout the later years of the patient's illness the symptoms of intestinal indigestion were very marked. The feces contained masses of undigested meat and starchy food, while the urine gave marked reactions for the products of intestinal putrefaction. The following urinary analysis is typical of all that were made. The volume voided in twenty-four hours could not be measured as incontinence was a symptom throughout the later years of the patient's life.

Urinary Analysis.—November 28, 1905. Color deep amber. Reaction very strongly acid. Sediment, heavy deposit of uric acid and urates, with many epithelial cells and a few leucocytes. Specific gravity, 1029. Urea, 2.25 per cent. Uric acid, 0.16 per cent. Ratio, $\frac{\text{urea}}{\text{uric acid}}$, 14.2. Nitrogen of ammonia, 2.9 per cent. of total nitrogen. Indican, marked reaction. Phenol, very marked reaction. Acetone, considerable amount. At no time was there a trace of albumin or any casts found in the urine.

The blood pressure was repeatedly taken with an old type of Riva-Rocci instrument. The blood pressure in the left arm was

constantly about 118. That in the right arm was higher, being about 170.

The examination of the heart and lungs was negative until the development of the terminal pneumonia.

The eyes were examined at intervals of about six months and there was at no time any evidence of optic neuritis. Two years before her death she began to develop deafness which was more marked in the right ear, but the hearing on the left side was also affected. The deafness began in September, 1904, and was described by the patient as occurring in rather definite cycles which commenced as a feeling of fulness in the ear, confusion of sounds and increasing deafness which by the end of fifteen minutes became marked. This condition remained at its height for an hour or more and then gradually subsided until the hearing became nearly normal. There was no tinnitus, no vertigo and no nausea. These symptoms would be repeated from three to five times daily, and at the end of about two weeks she noticed that the hearing did not fully return after the attacks. On October 18, 1904, she was examined by Dr. Alice E. Wakefield, whose report is as follows: "I found the left ear practically normal. The right showed slightly dull drum membrane. Tone limits were from 16 to 512; bone conduction diminished; Politzer acumeter heard six feet (perception low)." On November 25, Dr. Wakefield reported: "Right ear—Deafness markedly increased; acumeter 18 inches; no fork heard above 256; bone conduction could not be elicited; no nausea and no vertigo; tinnitus intermittent—not marked. The cycles which characterized the beginning of deafness have given way to permanent and rapidly increasing deafness. The left ear is still practically normal, although answers to tests are slowly and hesitatingly given. Eye grounds—Optic nerves are pale and arteries are small, but not exceeding physiological limits. The lesion involving the auditory nerve may lie anywhere along its course. It seems to me from the general symptoms that the lesion may be along that part of the nerve which lies in contact with the right lobe of the cerebellum at its junction with the anterior end of the worm, and could result from degenerative processes involving the right side of the cerebellum."

At Dr. Wakefield's request, an examination was also made

by Dr. E. Gruening, on December 2, 1904. He made the following report: "There was nothing positive in the ocular examination. I found both eyes perfectly sound. The affection of the right ear seems to have its seat in the labyrinth, inasmuch as no laterization to the affected ear could be obtained in the Weber test and Rinné was positive."

As the disease advanced there was a steadily increasing mental lethargy. The mentality became slow, but was always accurate. The patient's mind was very quickly tired. There were no perversions of the mental processes, apart from the dulling and slowing of thought and memory.

The following record was made on February 21, 1905, eighteen months before her death: She can stand, if balanced, and can walk if she is commanded to, and her attention is kept fixed. The legs are strong and show no atrophy. The hands and arms seem fairly normal, and a tremor which was once present in the right arm is rarely seen. The dynamometer is, for the right 40, for the left 35. There is no tremor of the face or legs, except at times; then it is coarse and intentional. She writes rather slowly and poorly; speech is slow. She cannot move in bed without help. The eye reflexes are normal, the kneejerks the same. Ocular excursions normal, except that there is a paralysis of convergence only. No forced laughter. Deglutition normal. There is deafness in the right ear and slight hyperesthesia of the right side of the face. There is no ataxia of the arms, and finger nose test is normal. There is no astereognosis. The patient had had sudden attacks in which she fell to the ground, without any distinct loss of consciousness or any convulsive movement. The attacks begin with pain in the occiput, which runs over to the front of the head, then she suddenly sinks to the ground.

On January 12, 1906, Dr. Wakefield again examined the eyes and ears and reported that the retinæ were pale, and the retinal arteries were small. There was a conjunctivitis probably trophic. The condition of the ears was the same as the year before.

The progress of the disease during the last six months of the patient's life was marked by a steady loss in muscular power and a gradually increasing mental hebetude. She could not stand or walk unsupported. Her head when she was resting in

her chair had a tendency to turn toward the left shoulder. In the later months she could not maintain an erect position in a chair or support her head. At this time, there were no cerebral palsies, except the deafness in the left ear. There was slight dulness to sensation on the right side of the cheek and tongue at times. There was very great slowness of deglutition and speech. No nystagmus or oculomotor palsy. No ataxia of the arms. The kneejerks were fairly good. There was transient rigidity of the arms and legs. There was a gradual failure of the memory—extraordinary slowness and retardation of thought, so that a reply would sometimes come out half a minute or one or two minutes after the question. She would start to express herself, then would lose the train of thought, which sometimes would come back to her later. There was no emotional disturbance, no headache or pain anywhere. She had no distinct paralysis of the limbs, but for months she was entirely helpless.

Two weeks before her death she had an attack of left hemiplegia, with clonus of both feet, and a very quick cerebral reflex to the plantar surface—like that of a baby, but no Babinski. There was a slight return of power following this attack, but an aspiration pneumonia developed which terminated fatally.

The autopsy was made by Dr. Libman and Dr. Israel Strauss. Their notes on the case are as follows:

Brain and Dura.—Dura, normal. Sinuses, normal. Brain, convolutions markedly flattened and fissures narrowed because of the condition of marked internal hydrocephalus. This was due to the pressure of a neoplasm lying on the upper surface of the left half of the cerebellum. The neoplasm was dark red in color, very vascular and in places showing psammomatous degeneration. It covered the entire left half of the cerebellum and encroached upon the worm. Posteriorly it was visible by its projection beyond the overhanging occipital lobe of the cerebrum. Anteriorly it reached as far forward as the corpora quadrigemina upon the left lobe of which it exerted considerable pressure. There was likewise a marked depression in the left crura cerebri and to a lesser degree in the cortex in the region of the hippocampal fissure. The left lobe of the cerebellum was reduced to almost half its thickness by the growth, but its substance was not infiltrated. The neoplasm was about 2 cm. in thickness, triangu-

lar in shape with its base posterior. It arose from the pia mater and nowhere showed any evidence of connection with the brain. Microscopical examination shows it to have been an endothelioma such as is characteristic of neoplasms arising from the pia.

Lungs: congestion at bases; in right lower lobe an area of purulent broncho-pneumonia, about 2 cm. in diameter.

Heart: brown atrophy with fatty degeneration; moderate atheroma of aorta; no congenital lesions.

Spleen: not enlarged, pulp soft, congested.

Liver: congested, fatty; gall-bladder and ducts negative.

Pancreas and adrenals: negative.

Kidneys: parenchymatous change, congestion; changes resulting from arteriosclerosis of the vessels.

Thyroid: normal.

In reviewing the history of this case, it will be seen that while the symptoms of brain tumor were not distinctive, those due to cerebellar involvement were marked throughout the course of the disease. These include the attacks of suboccipital headaches, the cerebellar ataxia, the cerebellar seizures—sudden relaxation and falling with no vertigo or unconsciousness—and the tendency when sitting to drop the head toward the left shoulder. The symptoms of involvement of cranial nerves also pointed to a localization of the disease at the base of the brain—the nerve deafness in the right ear, dysphagia and anesthesia of the tongue. But while the involvement of the cerebellum was evident, the lesion seemed to be one of degeneration rather than of compression from an extra-cerebellar growth.